withdrawal of sugar in the blood embarrassed the already damaged myocardium in this patient. Yet, faced with a marked ketosis and impending coma with so high an initial hyperglycemia, active insulin therapy was indicated. We did not anticipate such a marked response to the insulin in this case, a drop in blood sugar from 1,028 to 108 milligrams in seven hours, with a total of 290 units of insulin, especially with glucose administered hourly. The patient was permitted to maintain a blood sugar level higher than the renal threshold after her rapid response to insulin was observed. We are not familiar with the details of the death of this patient after her discharge from our direct care except that cardiac decompensation became acute and marked and death supervened within a short time.

P. O. Box HH.

REFERENCES

- 1. Foster, N. B.: Diabetic Coma, J. A. M. A., 84:719 (March 7), 1925.
- 2. Curtis, W. S., and Dixon, I. M.: Extreme Hyperglycemia in Diabetic Coma with Recovery, J. A. M. A., 90:1115 (April 7), 1928.
- 3. Shepardson, H. C., and Anderson, E. M.: Endocrinology, 13:188 (March-April), 1929.
- 4. Gray, P. A., and Sansum, W. D.: Diabetic Coma with Marked Hyperglycemia and Recovery, J. A. M. A., 97:230 (July 25), 1931.

FLEXIBLE ETHER MASK

By Harry S. Fist, M. D.

Los Angeles

THE flexible ether-mask frame here illustrated was devised by the author in an attempt to prevent traumatism of the face during anesthesia.

This frame is made of light-weight coil springs about five-sixteenths of an inch in diameter, one oval piece and two attached cross pieces as sketched, fastened together with thread or wire. The clean gauze covers which fold over the frame may be held with safety-pins or stitched with needle and thread. The result is a practically indestructible ether mask which is safe, flexible and adaptable to the face.

1930 Wilshire Boulevard.

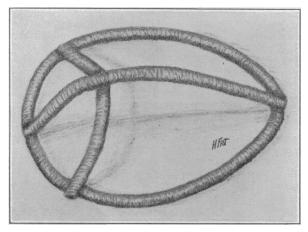


Fig. 1.—Drawing of flexible ether mask.

· CORONARY OCCLUSION WITH HYPERLEUKOCYTOSIS*

By Don Carlos Hines, M. D. San Francisco

REPORT OF CASE

ISTORY.—Mrs. E. M., age sixty-one, a negro cook, was admitted to Lane Hospital February 23, 1931, with the complaint of "pain over my heart" for three days. The family history was irrelevant. She had always been "strong and rugged."

Several years previously she had begun to notice slight precordial distress when fatigued, and mild palpitation and dyspnea on exertion. In 1930 she began a progressively downhill course but continued her work. A month before entry dyspnea and general weakness forced her to bed, and she called a physician, who prescribed "green drops," evidently digitalis. This did not benefit her, and she noted orthopnea and attacks of nocturnal dyspnea. She remained in bed most, but not all, of the time.

Three days before admission she was awakened at night by an excruciating nonradiating pain in the left upper chest accompanied by extreme dyspnea, nausea, and a sensation of smothering to death. The extreme pain lasted an hour, and a griping sensation persisted for several days. She vomited many times and had diarrhea. Two days before admission she began to cough up small amounts of blood. There had been a weight loss of thirty pounds in the preceding year.

Condition on Entry.—Physical examination on entry showed a thin, worn, dyspneic, moderately prostrated negress of sixty years. The temperature was 38.2 degrees centigrade rectally, the pulse 82, respirations 30. The blood pressure varied from 150/90 to 110/70. There was moderate general and retinal arteriosclerosis. The neck veins were distended when the patient was sitting up. There was a heaving apex impulse in the fifth intercostal space in the anterior axillary line. Heart sounds were faint and were obscured by an extensive friction rub. The pulse was regular but variable in force. The lung bases were dull, especially the right, with faint breath sounds and showers of crackles. The abdomen was somewhat distended, with resistance and some tenderness in the right upper quadrant. Liver edge and spleen were not felt. There was no edema and no general glandular enlargement. A clinical diagnosis was made of arteriosclerosis with degenerative heart disease and coronary occlusion.

Laboratory tests showed: blood Wassermann negative; hemoglobin 74 per cent Sahli; erythrocytes 4.2 million; leukocytes 140,000 with polymorphonuclear neutrophiles 74 per cent, lymphocytes 2 per cent and myelocytes 24 per cent. The erythrocytes varied moderately in size and shape, and many showed a deep polychromasia. Aside from their large number, there was nothing about the leukocytes to suggest leukemia. Almost all of the myelocytes were mature neutrophiles. A table of subsequent blood counts is appended.†

Urine and stool were normal. Electrocardiogram showed a rate of 75, sinus rhythm, normal intervals, left axis deviation, and inversion of T-waves in lead 1. In a roentgenogram of the chest (taken March 5) the measured cardiac area was 38 per cent above the predicted, and calcification was seen in the arch of the aorta.

Progress.—Her course in the hospital was characterized at first by slow improvement. The temperature continued slightly elevated, often touching 38, and once reaching 38.6 (rectally). The friction rub disappeared, allowing a musical systolic murmur to be heard over the precordium and vessels of neck and arms. There was no sputum.

The pulse began an upward trend on February 28, reaching 100 on March 2, when she noted an increase

 $[\]mbox{\ensuremath{^{\bullet}}}$ From the department of medicine, Stanford University Medical School.

[†] I am indebted to Dr. R. C. Mermod for checking some of the differential counts.